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CASE REPORT

Perivascular Cystic Degeneration of a Saphenous Vein Graft

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Introduction

Autogenous vein grafts are considered to be the best peripheral arterial replacement conduit with a patency rate of 44–86% after 5 years.[1,2] However, long-term patency of lower extremity bypass with autogenous venous grafts can be compromised by degenerative changes. Progressive atherosclerosis, fibrointimal hyperplasia, fibrosis and aneurysmal dilatation are the most important lesions responsible for the deterioration of the vein.[3–6] Here we present a case where the degeneration of a venous iliacopopliteal bypass finally resulted in an unusual large perivascular cyst.

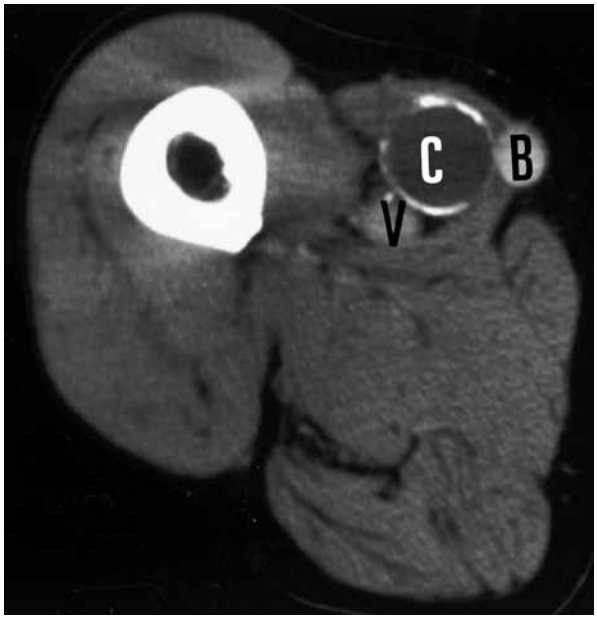

Fig. 1. CT scan demonstrating the superficial venous bypass (B), the adjacent large cyst (C) and the femoral vein (V). No contrast is seen in the cyst.

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Case Report

A 76-year-old man presented with a large mass in the right thigh. Sixteen years earlier he had undergone an ilio-popliteal bypass with the ipsilateral reversed saphenous vein because of severe intermittent claudication. Recovery was uneventful and the patient was discharged on the twelfth postoperative day. Since that time, however, the patient had been lost from follow-up. He now presented because of a mass in the right thigh, which had been present for about 5 years. It had been increasing in size progressively during the first 2 years of this period. It had never been painful. The patient used to be a heavy smoker (100 pack/years) but stopped 8 years previously after suffering a myocardial infarction. Arterial hypertension and age were the only current cardiovascular risk factors. On physical examination a 4×25 cm non-pulsatile mass was palpated on the anteromedial side of the right thigh. This longitudinally orientated mass was soft and painless and no thrill nor bruit was present. Both ankle pulses were present. There were no complaints of claudication, but chronic obstructive pulmonary disease prevented him from walking longer distances. Ultrasound revealed a 3 cm wide calcified tubular mass originating beneath the inguinal ligament and extending over 25 cm downwards to the medial part of the knee. On Doppler examination no flow was present in the mass. Ankle/arm index was 0.78. CT-scanning of the thigh demonstrated the tubular mass with a helical trajectory, adjacent to the venous bypass. Given the absence of contrast in the mass, no arterial

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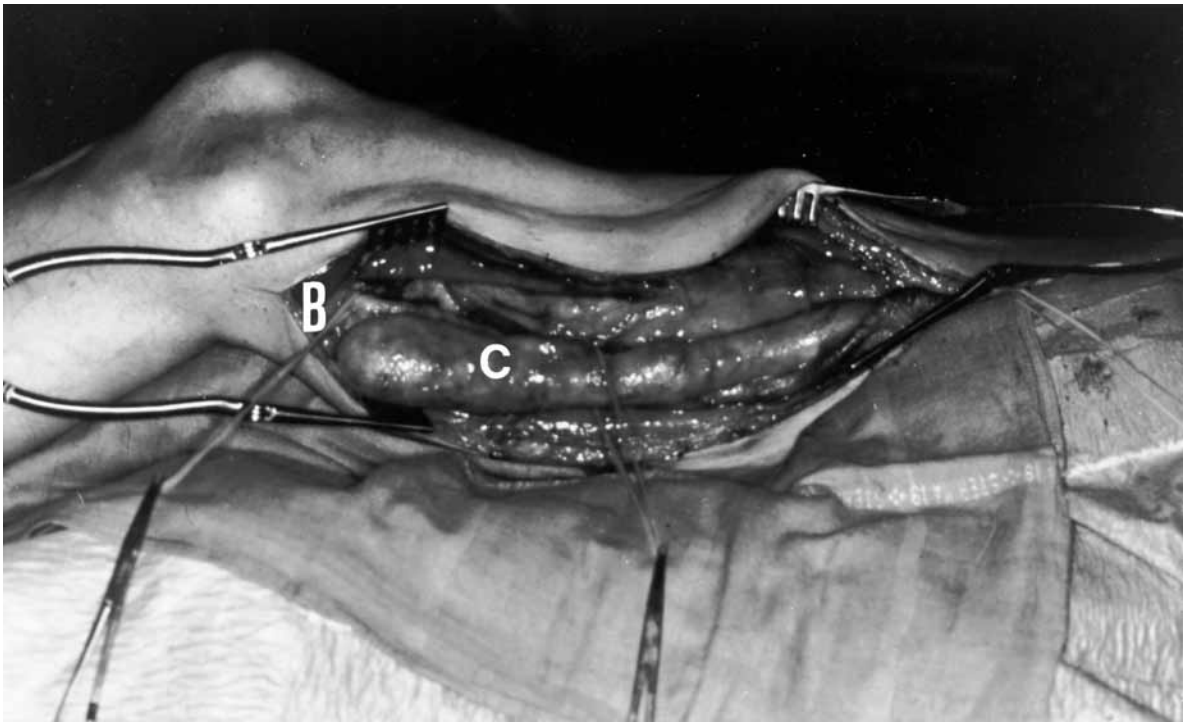

Fig. 2. Intraoperative view showing the large cyst from the groin to the knee (C) and the venous bypass graft (held in elastic band) (B).

communication seemed to be present (Fig. 1). Arteriography confirmed the patency of the iliacofemoral saphenous vein graft. Surgical exploration of the mass demonstrated a tubular cyst of 30 cm in length with a cross-section of 4 cm. The patent pulsating saphenous vein graft curled around this cyst like a helix (Fig. 2). Despite a careful search, no communication was found between graft and cyst. The cyst contained transparent serous fluid. The cyst was further carefully resected. It was impossible to remove the whole cyst without injury to the graft, so a small strip of cyst was left in place where it was attached to the graft. Postoperative recovery was uneventful. Macroscopic examination of the cyst showed marked atherosclerotic calcified plaques. Microscopic examination of the resected cyst wall defined its probable vascular origin, demonstrated by an inner regular lining with flat endothelial cells and a medial layer with the presence of muscular cells (Fig. 3).

Discussion

It is recognised that venous autografts undergo rapid structural changes, consisting primarily of marked thickening of the connective tissue in the vascular wall. The site of anastomosis is especially prone to degenerative changes, in particular to fibrointimal hyperplasia. Fibrointimal hyperplasia in venous grafts can be caused by endothelial changes due to damage during manipulation of the graft or ischaemia because of disruption of the vasa vasorum. Mechanical factors like increased shear forces and venous wall stress induced by arterial pressure or compliance mismatch may contribute to graft failure. Further, rapidly progressive vein graft atherosclerosis, which has been termed accelerated atherosclerosis, has been demonstrated from 6 months after implantation. Atherosclerotic aneurysmal degeneration of vein grafts is rare.[3–6] In our case histological examination revealed the probable vascular origin of the cyst. We presume a dissection of the venous conduit wall might have occurred underneath the inguinal ligament, progressing distally and stopping finally at the popliteal anastomosis. Repeated mechanical injury, due to flexion at the thigh, might have caused this dissection. However, the aneurysm contained only clear serous fluid and no contact with the lumen of the bypass could be found. The entry of the false lumen of the aneurysmal cyst might have closed over the years and the blood inside resorbed, resulting in a large perivascular cyst, without affecting the patency of the graft. To our best knowledge such a perivascular cyst probably resulting from dissection of an arterialised venous graft has not been described until now.

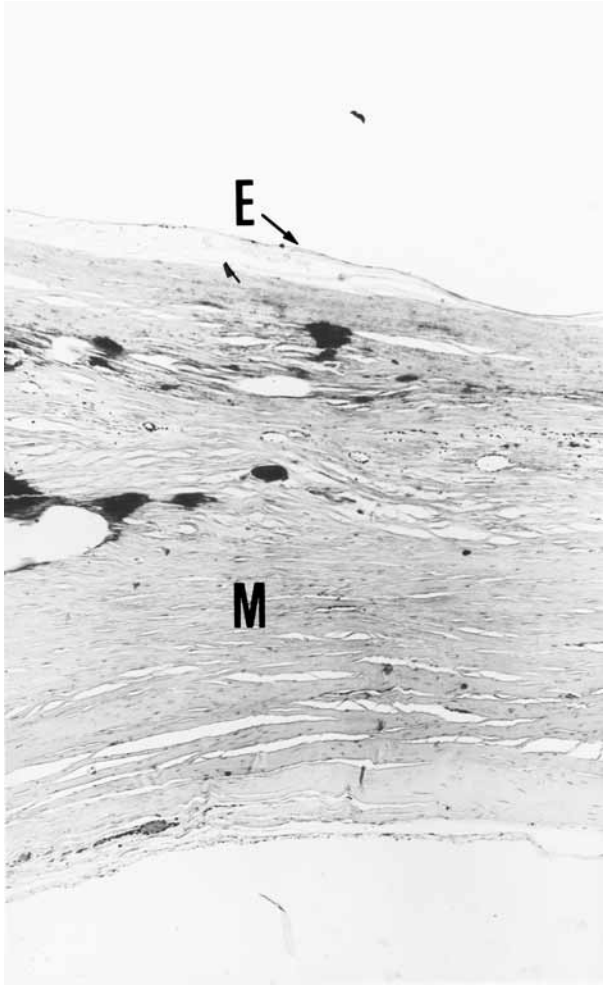

Fig. 3. Microscopy of the cyst wall demonstrating the endothelial lining (E) and the medial layer with the presence of muscular cells (M).

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